The mitochondrial abnormalities and enhanced mitophagy in the rhabdosphincter suggest that dysregulation of mitochondrial quality control may play a role in the development of urinary incontinence

Shinro Hata, Noriko Hamamatsu, Hironori Kai, Mayuka Shinohara, Hiromitsu Mimata, and Toshitaka Shin

Department of Urology, Oita University Faculty of Medicine, Japan

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Hypothesis / aims of study

In healthy striated muscle, damaged mitochondria are degraded through mitophagy, maintaining the function of striated muscle stem cells and muscle tissue homeostasis. While autophagy activity decreases with age leading to muscle mass reduction, detailed studies on mitochondrial morphology in the rhabdosphincter (RS) of elderly individuals are lacking. This study aimed to detect mitochondrial morphology and mitophagy activity in the RS.

Study design, materials and methods

To investigate the mitochondrial abnormalities,morphological observations of mitochondria were performed using transmission electron microscopy (TEM). Next, the expression of mitophagy-related factors was analyzed by reverse transcription-polymerase chain reaction (RT-PCR).

Results

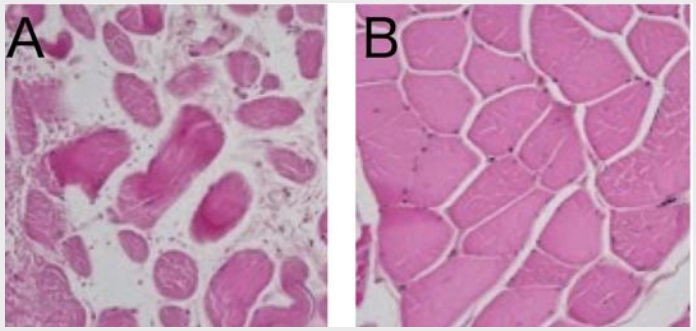

Fig 1 Hematoxylin and eosin stain

A; RS
B; RA

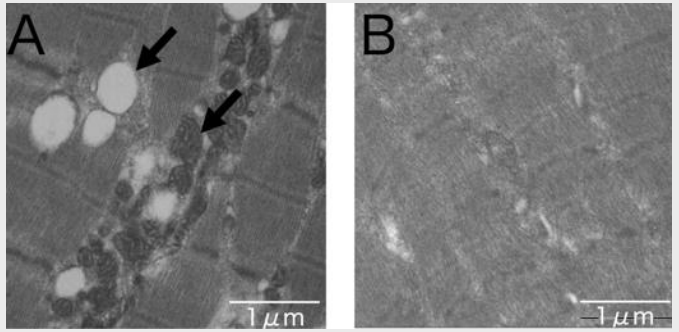

Fig 2 TEM

RS had more gaps in the muscle bundles, fewer mitochondria, and structural anomalies compared to RA.

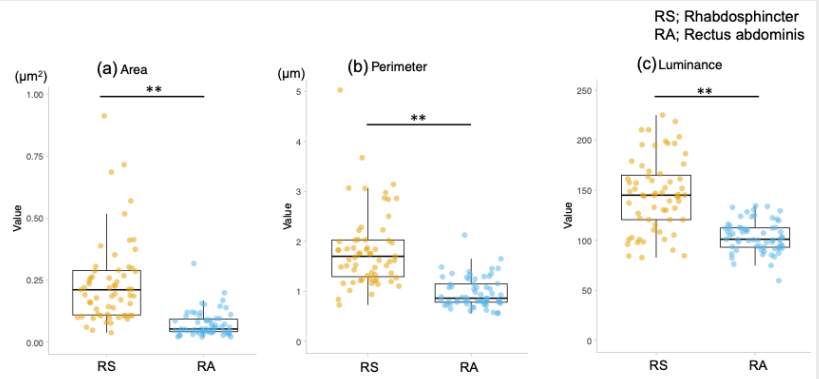

Fig 3 A quantitative analysis of mitochondrial morphology

The area, perimeter, and luminance of mitochondria in the RS tissue were greater in size, longer in perimeter, and higher in luminance compared to the RA tissue, respectively ($p < 0.01$).

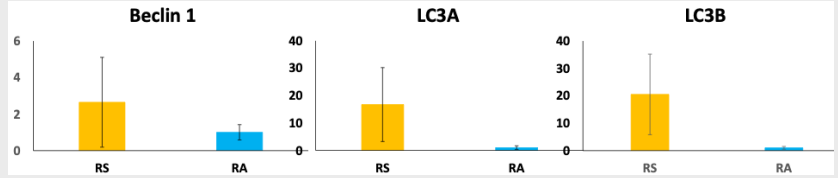

Fig 4 Analysis of mitophagy factors by RT-PCR

RT-PCR analysis showed enhanced expression of LC3A/B and Beclin1 in RS tissue, indicating enhanced mitophagy activity.

Conclusions

The findings indicate significant mitochondrial morphological abnormalities and enhanced mitophagy in the RS tissue compared to the RA tissue. These changes suggest increased mitochondrial damage and turnover in the RS, potentially contributing to functional decline.